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A Case of Tumor Lysis Syndrome in a Patient with Pancreatic Adenocarcinoma Treated with Low-Dose Gemcitabine

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ABSTRACT

Background: Tumor lysis syndrome (TLS) is a common adverse consequence of treatment of high-grade hematologic malignancies that has been known to occur rarely in some solid tumors, including small cell lung cancer, breast cancer, colorectal cancer, neuroblastoma, ovarian cancer, and hepatocellular carcinoma.

Case Report: We present a case of TLS in a patient with pancreatic adenocarcinoma treated with one small dose of gemcitabine. To our knowledge, this phenomenon has only been described once prior in the medical literature and never with a reduced dose of chemotherapy.

Conclusion: This case reveals the need for heightened awareness of TLS in patients with solid tumors, especially in patients with pancreatic adenocarcinoma.

Keywords: *Adenocarcinoma, gemcitabine, tumor lysis syndrome*

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INTRODUCTION

Tumor lysis syndrome (TLS) is a common adverse consequence of treatment of high-grade hematologic malignancies that has been known to occur rarely in some solid tumors, including small cell lung cancer, breast cancer, colorectal cancer, neuroblastoma, ovarian cancer, and hepatocellular carcinoma.[1-3]

TLS is caused by massive tumor lysis with the release of intracellular contents into the bloodstream, including potassium, phosphorus, and nucleic acids that are then degraded into uric acid. These contents are filtered by the kidneys and cause renal vasoconstriction and inflammation, leading to acute kidney injury.[2] Hyperkalemia can precipitate cardiac arrhythmias and sudden death. TLS is an emergency that must either be prevented or recognized and treated early in its course to prevent kidney failure and sudden death. The mortality of TLS is higher in patients with solid tumors, likely because of its lower incidence and therefore lack of prophylaxis.[4]

To our knowledge, only one case of TLS has been reported after treatment of pancreatic adenocarcinoma. We present the first known case of tumor lysis to occur after one half-dose administration of gemcitabine.[5]

CASE REPORT

A 68-year-old male with well-controlled diabetes mellitus, hypertension, and recently diagnosed, biopsy-proven, stage IV pancreatic adenocarcinoma with metastases to the liver (Figure) and poor functional status presented to the emergency department 5 days after receiving his first and only treatment of 500 mg/m^2 (half the usual dose of 1,000 mg/m^2) of gemcitabine. The patient reported weakness, dyspnea on exertion, lower extremity swelling, and poor oral intake during the previous 3 days. He had a potassium level of 6.8 mmol/L, phosphorus of 11.2 mg/dL, uric acid of 20.9 mg/dL, blood urea nitrogen of 181 mg/dL, and creatinine of 6.02 mg/dL. His hemoglobin concentration was low at 8.3 g/dL; however, his baseline hemoglobin was 8.8 g/dL, and workup at the time had indicated anemia of chronic disease. His platelet count had dropped from 312,000/mcL to 65,000/mcL; however, atypical hemolytic uremic syndrome was ruled out because of the patient's high haptoglobin level and normal lactate dehydrogenase level and a peripheral smear that showed no schistocytes. The smear did not indicate hematologic malignancy; no blasts or other immature cells were found. Rhabdomyolysis was not considered as a diagnosis because the patient's liver transaminases were not elevated.

A diagnosis of TLS was made, and the patient was put on rasburicase and allopurinol for hyperuricemia, given bicarbonate infusion and sodium polystyrene for hyperkalemia, and administered sevelamer carbonate for hyperphosphatemia. The patient's electrolyte abnormalities resolved

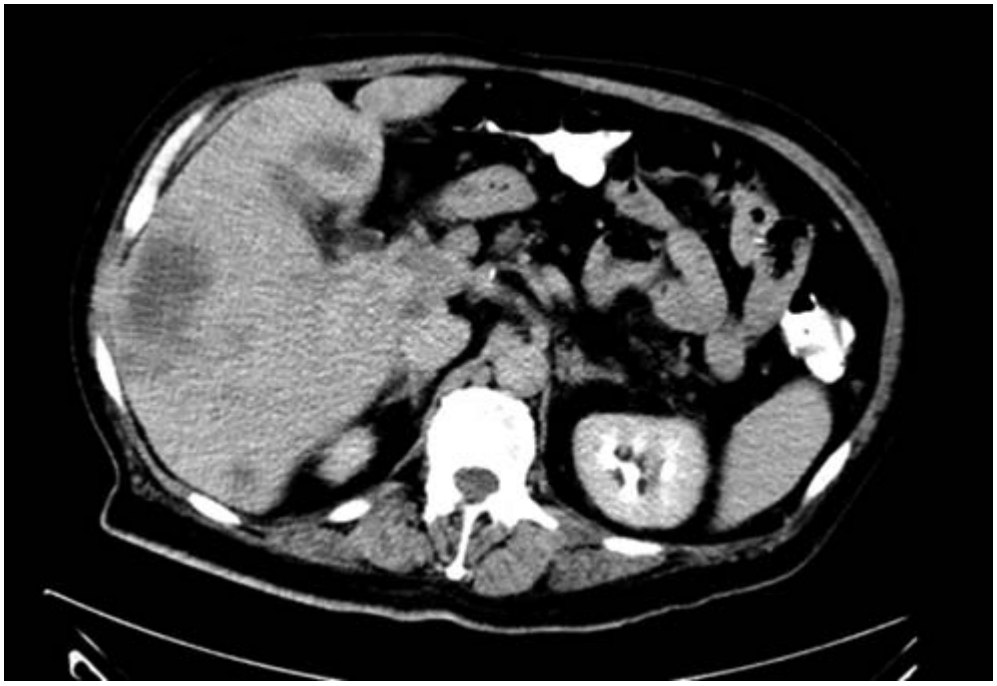

Figure. Computed tomography of the abdomen on admission demonstrates a large tumor burden in the liver.

during a period of 3 days, requiring only one dose of rasburicase.

He was transferred out of the medical intensive care unit in stable condition. Review of his history revealed a baseline creatinine level of 1.13 mg/dL (glomerular filtration rate >60 mL/min/1.73 m^2), hemoglobin A1c of 5.6%, and a potassium level of 4.1 mmol/L. He had no history of gout or hyperuricemia. His carbohydrate antigen (CA) 19-9 level was 15,082 U/mL, carcinoembryonic antigen was 1.1 ng/mL, and chromogranin A was 6.6 ng/mL. His dose of gemcitabine was reduced because of his limited functional capacity. He had been able to perform his activities of daily living; however, he could not ambulate independently, a likely consequence of his poor oral intake because of his large abdominal tumor burden.

After the patient stabilized, he declined further chemotherapy and accepted hospice care. He continued to be intolerant of oral nutrition and refused artificial nutrition. He died 14 days later. No follow-up imaging or CA 19-9 levels were obtained.

DISCUSSION

TLS is a serious consequence of chemotherapy that must be recognized early and treated aggressively to avoid renal failure and death. This case is important because it sheds light on the capability of a solid tumor such as pancreatic adenocarcinoma to lyse with a diminished dose of chemotherapy in such a manner as to lead to TLS. This possibility may indicate that TLS is more common than we believe and that prophylaxis for TLS may be indicated in some aggressive solid tumors.

CONCLUSION

The threshold for suspecting TLS in solid tumors must be lowered to avoid preventable deaths. Observational studies are needed to study the indications for solid tumor TLS prophylaxis.

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